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Letter to the Editor

Helicobacter pylori's potential association with epilepsy

Dear Editor,

We have read with interest the study by Asadi-Pooya et al. who concluded that the rate of *Helicobacter pylori* infection (*Hp*-I) was not higher in patients with epilepsy compared to healthy individuals.[1] However, due to problems mainly encountered during recruitment of the patients and controls of this study, the conclusion of this trial might be not reasonable.

It has been reported that the prevalence of *Hp*-I in the city of Shiraz, where the study by Asadi-Pooya et al. was conducted, is very high,[2,3] regardless of the socioeconomic status, an already established significant factor affecting *Hp* prevalence in the European countries. This means that to prove a difference in *Hp* prevalence between any two groups in Shiraz would require several hundreds or even a few thousands of participants. Instead, Asadi-Pooya et al. presented their results based on a small number of patients and controls (34 and 33, respectively) and therefore the power of their study was too low. Specifically, the study groups were not age-matched. It seems that the control group was about 10 years older than the idiopathic generalized epilepsy (IGE) group and 2–3 years older than the temporal lobe epilepsy (TLE) group. *Hp* prevalence is higher in older individuals, and this explains why *Hp*-I was detected in 72.7% in the control group compared with 61.8% in the IGE and 50% of the TLE group. These differences did not reach a statistical significance due to the very small number of patients recruited, but it is almost certain that the *p* value equal to 0.068 reported would have been significant if the authors had recruited just a few more patients. In that case the control healthy group would have a higher *Hp* prevalence than the epileptic patients, leading to the paradox conclusion that *Hp* might protect against epilepsy.

On the other hand, recent data suggest a probable association between *Hp*-I and epilepsy, especially with poor prognosis.[4] In this respect, we speculate that *Hp*-I, by inducing proinflammatory cytokine production and blood–brain barrier (BBB) disruption,[5] also mentioned by the aurhors,[1] may lead to neuroinflammation and neuronal damage in epilepsy thereby triggering seizures' induction and epilepsy progression.[5]

Conflict of interest

None.

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